Superoxide Production in PMNs from Leprosy Patients

TO THE EDITOR:

In a recent paper (Int. J. Lepr. **46** [1978] 337–441), Dr. O. Rojas-Espinosa reported his results in determining superoxide production of polymorphonuclear leukocytes (PMNs) from patients with leprosy as compared with those from normal individuals. Levels obtained were essentially similar. In addition, he found no significant difference between superoxide production of PMNs from patients with standard lepromatous leprosy and that of PMNs from patients with reactional leprosy (RLL). The author analyzed his results and compared them with those previously reported by us (Clin. Exp. Immunol. **20** [1975] 257–264) as follows: "Goihman-Yahr, *et al.*, found that patients with any type of leprosy, except reactional (RLL) lepromatous leprosy, had normal numbers of NBT-reducing cells. In patients with RLL, the proportion of reducing cells was significantly raised. We did not find a significant increase in the O_2^- levels produced by PMN from patients with RLL when compared with lepromatous patients without reaction."

From these comments, the reader might conclude that Dr. Rojas' results are at variance with ours, at least concerning RLL. This is not the case at all. As I feel that PMN activation is a rather distinctive feature of RLL, the issue should be clarified.

In the method which we used (a modification of Matula and Paterson's, New Engl. J. Med. **285** [1971] 311–317), heparinized peripheral blood is incubated with NBT at 37°C in siliconized excavated glass slides. We found that blood from patients with active RLL had a significantly higher proportion of NBT-reducing PMNs than blood from normal individuals or from any other kind of leprosy patients. We also found that the above was not due to any intrinsic difference between PMNs from RLL patients and those from other persons. Thus, if blood was incubated *in vitro* with endotoxin and NBT, the proportion of NBT-reducing PMNs reached a similarly high level in all groups. We concluded that spontaneous activation (i.e., without incubation with an additional activator) was brought about in RLL patients by some factor, presumably of immunologic nature. Further work has been done in this direction, but it is not germane to the current discussion.

Dr. Rojas-Espinosa employed a method by which PMNs were isolated from peripheral blood and then put in the cold (thereby presumably suppressing any pre-existing metabolic burst). PMNs were then incubated at 37°C with cytochrome and latex particles. The latter are quite capable of inducing an activation comparable to that caused by endotoxin. Dr. Rojas was then

simply determining the capacity of PMNs to become activated *in vitro* and to generate superoxide. He did not find out whether activation pre-existed in patients with RLL. His results are concordant with ours except that he did not explore what was happening to the metabolism of PMNs in the patients when they had symptomatic RLL. This latter point is a key one in our concept. It must be added that it is conceivable that NBT reduction, as estimated by cytologic methods, is affected not only by true metabolic activation but also by availability of NBT to the cell. Factors such as PMN permeability may be of importance.

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